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Model for Measuring the Health Impact from Changing Levels of Ambient Air Pollution

Morbidity Study

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Based on the significant associations between the pollutants and the disease groups, holding climatological and days-of-week variables constant, the variation due to the pollutant is estimated. According to the results, sulfur dioxide based on patient exposure levels can account for about 13% of the variation of emergency room visits for acute bronchial and lower respiratory infections and about 22% for total cardiac diagnoses. Nitric oxide based on measurements from the closest site to the hospitals can account for about 7% of the variation of visits for total respiratory diagnoses, 6% for allergic conditions and upper respiratory infections, 4% for total cardiac diagnoses and 4% for hypertension and vascular heart diseases. Total suspended particulate, carbon monoxide and ozone do not show significant associations with any disease groups.

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by

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis, and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the development and revision of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for noniodizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

The report examines the association of day-to-day variation in ambient pollution concentrations and morbidity as measured by emergency room visits for cardiac and respiratory diseases in two major hospitals in the City of Chicago. This is the morbidity phase of a research project to quantitate the association between health effects and ambient pollutant concentrations in a major metropolitan area.

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Director
Health Effects Research Laboratory

ABSTRACT

This study quantitatively examines the relationship between human health and ambient air concentrations of the major pollutants in the city of Chicago. The study consists of two major parts; a morbidity component and a mortality component. This report describes the morbidity analysis in which linear regression models have been developed to quantitatively estimate the degree of the air pollution contribution to emergency room visits for cardiac and respiratory diseases in two major hospitals in the city of Chicago.

Data on emergency room patients were collected on every Tuesday, Wednesday and Thursday from April 12, 1977 to April 14, 1978. Air measurements used in the study are total suspended particulate, sulfur dioxide and nitrogen dioxide from 22 sites of the Chicago Air Sampling Network (CASN). Also used are measurements of sulfur dioxide, nitrogen dioxide, nitric oxide, carbon monoxide and ozone from the Illinois E.P.A. monitoring site on Polk Street at the University of Illinois Medical Center, the site closest to the hospitals. Daily climatological measurements used are from Midway Airport, as published by the U.S. Department of Commerce.

Based on pollution measurements (TSP, SO_2 and NO_2) from CASN and the patient's home address, exposure levels were estimated for each of the 21,000 patients. A daily average exposure level was obtained by dividing the sum of the individual exposure levels by the number of patients in a specific disease category. With the pollution measurements from Polk Street, one reading was used as an alternate of an average exposure index.

A dependent variable in multiple regression analysis is the percentage of excess visits for each disease group, which is a ratio of the number of visits to the average number of visits in a specific disease group, expressed as a percentage. Three types of independent variables are used; air pollutants, climatological measurements and days of the week as dummy variables. Only one pollutant is included in each equation, since the number of observations differs from one pollutant to the next. Based on the significant associations between the pollutants and the disease groups, holding climatological and days-of-the-week variables constant, the variation due to the pollutant is estimated.

According to the results, sulfur dioxide based on patient exposure levels can account for about 13% of the variation of emergency room visits for acute bronchial and lower respiratory infections and about 22% for total cardiac diagnoses. Nitric oxide based on measurements from the closest site to the hospitals can account for about 7% of the variation of visits for total respiratory diagnoses, 6% for allergic conditions and upper respiratory infections, 4% for total cardiac diagnoses and 4% for hypertension and vascular heart diseases. Total suspended particulate, carbon monoxide and ozone do not show significant associations with any disease groups.

This report was submitted in fulfillment of Contract No. 68-02-2492 by the University of Illinois School of Public Health, under the sponsorship of the U.S. Environmental Protection Agency. This report covers a period from February 15, 1976 to October 15, 1978, and work was completed as of December 31, 1978.

CONTENTS

	Page
Disclaimer	ii
Foreword	iii
Abstract	iv
Figures	vii
Tables	viii
Acknowledgement	x
1. Introduction	1
2. Conclusions	2
3. Recommendations	3
4. Background Information	4
The Problem	4
Studies in Chicago	5
5. The Method	9
Source and Collection of Data	9
Hospital Data	9
Climatological Data	11
Air Pollution Data	18
Estimation of Missing Pollution Measurements	22
Calculation of Community Area Exposure Levels	24
Calculation of Disease Specific Average Exposure Levels	29
Multiple Regression Analysis	29
6. Results and Discussions	33
Models for Total Respiratory Diagnoses (TRD)	33
Models for Allergic Conditions and Upper Respiratory Infections (RD1)	36
Models for Acute Bronchial and Lower Respiratory Infections (RD2)	36
Models for Total Cardiac Diagnoses (TCD)	39
Models for Hypertension and Vascular Heart Disease (CD1)	41
The Variation Due to the Pollutant, the Days-of-Week Effects and Weather Changes	43
Comments	46
References	48
Appendix	51

FIGURES

		Page
1.	Location of air monitoring sites and 76 community areas in Chicago	19

TABLES

		Page
1.	List of cardiac and respiratory diagnoses	12
2.	Daily Means of Emergency Room Visits for Major Disease Groups	17
3.	City of Chicago, Department of Environmental Control-Chicago Air Sampling Network	21
4.	No. of Days Monitoring Station Shutdown	23
5.	Predication Equations for TSP missing data	25
6.	Alternate equations for TSP missing data	26
7.	Prediction equations for SO_2 missing data	27
8.	Prediction equations for NO_2 data	28
9.	Morbidity Models: Total Respiratory Diagnoses (TRD)	34
10.	Morbidity Models: Allergic Conditions and Upper Respiratory Infections (RD1)	37
11.	Morbidity Models: Acute Bronchial and Lower Respiratory Infections (RD2)	38
12.	Morbidity Models: Total Cardiac Diagnoses (TCD)	40
13.	Morbidity Models: Hypertension and Vascular Heart Disease (CD1)	42
14.	The variation due to the pollutant, days-of-week effects and weather changes in the models selected from Tables 9-13	44
15.	Correlation coefficients between emergency room visits for cardiac and respiratory conditions and environmental measurements in phase I analysis	52
16.	Correlation coefficients between emergency room visits for cardiac and respiratory conditions and environmental measurements in phase II analysis	53
17.	Correlation coefficients between emergency room visits for cardiac and respiratory conditions and environmental measurements in phase III analysis	54

TABLES (continued)

		Page
18.	Correlation coefficients between emergency room visits for cardiac and respiratory conditions and environmental measurements in phase IV analysis	55
19.	Correlation matrix of environmental measurements in phase I analysis	56
20.	Correlation matrix of environmental measurements in phase II analysis	57
21.	Correlation matrix of environmental measurements in phase III analysis	58
22.	Correlation matrix of environmental measurements in phase IV analysis	59
23.	Distribution by race of study population; April, 1977-April, 1978	60
24.	Distribution by sex of study population; April, 1977-April, 1978	60
25.	Frequency age distribution of total study population, CCH and UIH, April, 1977-April, 1978	61
26.	Frequency of all diagnostic illnesses of study population, April, 1977-April, 1978	62

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SECTION 1

INTRODUCTION

The United States Congress passed the Clean Air Act in 1967 and its Amendments in 1970 to protect and enhance the quality of the nation's air resources while promoting the public health and welfare and the productive capacity of its population, reducing harmful emissions, and ensuring that air pollution problems will, in the future, be controlled in a systematic way. Also, in 1970 the city of Chicago passed an ordinance which virtually banned coal and garbage burning in individual households and businesses.

Such legislative efforts to control air pollution had significant effects on a decrease in the amount of some pollutants in the city of Chicago; suspended particulate levels had dropped 26 per cent, and sulfur dioxide levels had been cut by 50 per cent between 1970 and 1975. However, carbon monoxide and ozone levels have remained high, in spite of Chicago's voluntary auto emissions control program.

The present study has quantitatively examined the relationship between human health and ambient air concentrations of the major pollutants in the city of Chicago. The study has consisted of two major parts; a morbidity component and a mortality component. This report describes the morbidity analysis in which linear regression models have been developed to quantitatively estimate how air pollution affects the number of emergency room visits for cardiac and respiratory diseases in two hospitals in the city of Chicago.

SECTION 2

CONCLUSIONS

A total of forty regression models were developed to examine the strength of the relationship between an individual pollutant and cardiac and respiratory morbidity, controlling for climatological and days-of-week variables. In spite of limited data base on ambient air pollution measurements, it is likely that air pollution accounts for some portion of the variation in the incidence of cardiac and respiratory morbidity. The following conclusions were made:

1. Sulfur dioxide based on patient exposure levels has explained about 13% of the variation in emergency room visits for acute bronchial and lower respiratory infections, and about 22% of the variation in emergency room visits for total cardiac diagnoses in Cook County Hospital and the University of Illinois Hospital combined.
2. Nitric oxide based on measurements from the closest site to these hospitals explained about 7% of the variation in visits for total respiratory diagnoses, 6% of the variation in visits for allergic conditions and upper respiratory infections, 4% of the variation in visits for total cardiac diagnoses and 4% of the variation in visits for hypertension and vascular heart diseases.
3. Nitrogen dioxide based on measurements from the closest site to these hospitals explained only less than 1% of the variation in visits for total cardiac diagnoses.
4. Total suspended particulate, carbon monoxide and ozone have not shown significant associations with any disease groups.

SECTION 3

RECOMMENDATIONS

The most critical limitation in the present study was the lack of reliable daily measurements of ambient air pollutants throughout the city of Chicago. It is strongly recommended that the re-evaluation of the air pollution monitoring systems in Chicago be conducted as soon as possible and more accurate, reliable and usable pollution measurements be available to users, especially for purposes of epidemiological research.

Air pollution measurements at only Polk Street,the closest site to Cook County Hospital and the University of Illinois Hospital, were used as the alternate of the average patient exposure levels because they were the only available data at the time of the data collection. Air pollution measurements from other sites should be sought in the future to measure patient exposure levels more accurately than the site at Polk Street. The models developed in this study can be refined and validated only if a sufficient number of observations are made of reliable ambient air pollution measurements from different monitoring sites covering the entire Chicago area.

Emphasis should be placed on the following if meaningful models are to be developed: (1) vastly improved air pollution data base, (2) extension of collection period to span a number of years for seasonal analysis, (3) development of a seasonal model for other pollutants besides ozone, (4) re-grouping disease diagnoses to examine acute effects of each pollutant, and (5) examining lag-effects after obtaining more pollution measurements from other sites in addition to those at Polk Street.

SECTION 4

BACKGROUND INFORMATION

THE PROBLEM

Extremely polluted air is known to be a killer of old people with chronic illnesses and persons with serious illnesses, as shown in episodes in the Meuse Valley[1], Donora[2], London[3], and New York City[4].

In addition to such an acute effect, long term exposure to aggravated air may cause or exacerbate chronic respiratory diseases, as reported in the Tokyo-Yokohama area[5-10], New Orleans[11], and Yokkaichi City[12,13]. In all of these cases, the air pollution level was significantly h gher than the usual level found in most urban areas.

At usually prevailing levels of air pollution, on the other hand, direct relationships may not be expected between diseases and daily levels of air pollution by using a simple epidemiological approach in which frequency distribution of diseases is compared with levels of air pollution. Multiple regression analysis is one of the methods to obtain quantitative estimates of the relationship between diseases and environmental factors. Multiple regression methods had been used to examine the degree of air pollution's contribution to mortality for the past decade by some researchers like Sprague and Hagstrom[14], Lave and Seskin[15,16], Hodgeson[17], Buechley and his co-workers[18], and Schimmel and Murawski[19]. In particular, Lave and Seskin[20] have carried out an extensive study to quantitatively measure air pollution effects on mortality. They estimated that a 50 percent reduction in sulfates and suspended particulate would account for a 4.7 percent decrease in the unadjusted total mortality rate, based on their 1960 annual cross-sectional analysis.

While many regression analyses had been done by using mortality data in relation to air pollution, climatological measurements and socioeconomic variables, air pollution effects on morbidity had been estimated in terms of a dose-response relationship using regression analysis, by only a few researchers. Thompson and his co-workers[21] examined relative contributions of air pollution to morbidity over a period of nine consecutive seasons by using the stepwise regression method. Their results showed that the regressions differed markedly by season of the year, and that the climatological

variables seemed more related to common cold rates than the air pollutants.

STUDIES IN CHICAGO

For the past ten years, the Chicago Air Pollution Study Group has been examining the health effects of air pollution, particularly, to define those individuals in the pollutants at which their health is adversely affected.

The Chicago Chronic Bronchopulmonary Disease Registry Study[22] began in August, 1966. A total of 571 patients from 16 different facilities, chiefly, pulmonary and allergy clinics, were gathered into a registry. They were classified according to the severity of disease and each maintained a daily record of acute chest illness. Using the continuous air pollution monitoring network of the city of Chicago, a model was developed to assign an air pollution level (SO_2 and particulates) to each square mile of the city for every 15 minutes of the day. Patients' exposures were determined by the estimated level of pollution in the residence and occupation for each 24 hour period. Illness appeared to be correlated with levels of sulfur dioxide with increasing illness at each of 7 levels of SO_2 pollution. At 0.24 ppm for 24 hours, there were more than twice as many acute chest illnesses as when the level was 0.04 ppm. This occurred in males 55 and over with advanced bronchitis and to a lesser degree in those under the age of 55 with similar bronchitis severity. Those with no bronchitis or mild bronchitis showed no effect at these levels. Further analysis, which compared the number of sick to well days for each individual in relation to his air pollution exposure, showed a significant relationship in two of three months examined for the same age groups. It appeared that when SO_2 was considered as a pollution index in males 55 and over with advanced bronchitis, there was a relationship between levels of pollution and frequency of acute chest illnesses.

An additional group of subjects consisted of 51 patients followed in a chronic respiratory disease clinic[22]. In addition to being classified according to disease severity, each patient maintained a daily record of increase or decrease in coughs and dyspnea, and also was trained to examine his sputum every morning. If the sputum was purulent, the individual was instructed to start on a broad-spectrum antibiotic on which he remained for

at least seven days. The development of purulence and the use of antibiotics were also recorded. The daily record shown in the form of a postcard was returned each week. Then, each patient's exposure level to SO_2 was estimated by using daily SO_2 measurements from the air monitoring network in Chicago. All patients were classified into three groups: (1) 0-0.09 ppm, (2) 0.10-0.19 ppm, and (3) 0.20 ppm and over. Acute illness (increasing purulent sputum, cough, and dyspnea) was reported in 35% of patients at 0.20 ppm and over, 24% at 0.10-0.19 ppm, and 23% at 0-0.09 ppm in October, 1967. The same tendency was observed in November and December, 1967. It was concluded that exacerbation of symptoms and precipitation of acute respiratory illness occurred at high daily levels of SO_2 exposure among elderly persons with advanced bronchitis.

Lepper and his associates[23] examined deaths from all respiratory causes, including tuberculosis and lung and bronchial cancer, in the total white population of the city of Chicago in the years 1964 and 1965 by pollution levels and socioeconomic status. Census tracts were classified socioeconomically and placed in pollution categories based on annual averages of sulfur dioxide. The study utilized SO_2 values measured at 21 stations spread over the city during three 24 hour periods weekly. Annual average isopleths were drawn from the data for approximately 150 days in which measurements were made and an average annual exposure concentration was calculated from these for each census tract. Census tracts were divided on socioeconomic criteria into high, medium, and low. Each of these three subpopulations were classified into three groups: (1) the highest level of SO_2, (2) the moderate level, and (3) the lowest level - according to annual exposure concentrations. Only the white population was analyzed because of the lack of a high socioeconomic group in the nonwhite population. The results revealed a significant rise in the total respiratory death rate from high to low socioeconomic groups in relation to levels of SO_2. At the low SO_2 level, significant differences were not present between classes; while at the moderate SO_2 level, the rate of the low socioeconomic cell was significantly higher than those of the medium and upper socioeconomic groups. This was also true when they were compared at high SO_2 levels. The groups with the highest death rates were the lowest socioeconomic groups exposed to the highest pollution levels.

Carnow and Feiveson[24] examined the acute effects of SO_2 on mortality in the city of Chicago during a nine day episode (October 26 through November 3, 1969). Because SO_2 concentrations varied widely throughout the city during the episode, the city was divided into three sections (the high SO_2 section, the moderate, and the low) according to the average SO_2 concentration during the episode at the TAM station or stations closest to the area in which the decedent resided. The sharpest increases in mortality from cardiac disease occurred in males, mainly older males, who are known to be most susceptible to cardiac difficulties. Furthermore, these increases occurred only in the areas of the city with highest average pollution levels during the nine days of the episode. Some striking increases in mortality were also observed in certain subgroups of females in the high pollution areas; for example, there were 16 deaths during the episode from rheumatic heart disease in white females as compared to 8 in the nine days before, 8 following, and 12 expected. In the moderate pollution section, there was also a substantial increase in ischemic heart disease mortality for white females; 72 deaths during the episode, 48 deaths before, 55 after, and 54 expected. There were no significant increases in mortality in the low pollution areas during the episode as compared to before and after the episode.

In the Drexel Home Study[25], 49 normal volunteers in a home for the aged were studied. They were classified according to severity of bronchitis, and each person was examined at the same time each day, Monday through Friday for a total of 25 days. At each visit, resting pulse, blood pressure, and respiratory rate were obtained along with responses to questions regarding symptoms relating to cardiovascular, respiratory, and gastrointestinal systems. Spirometer measurements were carried out using a Jones Pulmonar, 6-liter bellows dry spirometer. Determinations were then made of the one-second, three-second, and total force expiratory volume and maximum expiratory flow rate. In addition, peak flow was measured using a Wright Peak Flow meter. Each member of the study team was assigned his own panel of patients and performed the initial history, physical examination, daily recording of symptoms, and pulmonary function. Variations in levels of SO_2 and particulate were compared with daily spirometry and peak flow in the individuals in the study. The result

suggested no relationship between the levels of SO_2 and particulate and pulmonary function in this population during the period of the study. Other analyses also revealed no significant changes in function or symptoms. While the mean levels of SO_2 during the study were relatively low for Chicago, 0.024 ppm, the mean particulate level was 165 $\mu g/m^3$ with a range of 72 to 309 µg, levels at which health effects were noted in some segments of the population in a number of other studies. The results suggest that this nonsmoking urban aged population represented a hardy survival population.

With the cooperation of the Chicago Lung Association and fourteen hospitals throughout the city of Chicago, Namekata and Carnow have carried out a study to examine effects of air pollution on the incidence of cardiac and respiratory diseases by using individual records of emergency room visits in those hospitals[26]. Recently, they have re-examined effects of sulfur dioxide and total suspended particulate on cardiac and respiratory conditions, based on the same morbidity data and estimated patient exposure levels to those pollutants[27]. Their regression model for all respiratory conditions included sulfur dioxide, daily maximum temperature and daily average wind speed as independent variables of which all F statistics are significant. The model for bronchitis included total suspended particulate, daily minimum temperature, daily average wind speed and humidity which indicated all significant F statistics. These findings are meaningful because statistical significance of the partial regression coefficient of each pollutant implies that such a pollutant may possibly have critical impact on the disease even after partitioning the effects of other independent variables included in a equation. However, their models for asthma and for cardiac conditions did not show significant results.

SECTION 5

THE METHOD

SOURCE AND COLLECTION OF DATA

Hospital Data

The two hospitals used as the source of morbidity patient data were Cook County (CCH) and the University of Illinois **Hospitals (UIH).** CCH was chosen as the initial collection site for three reasons. First, in a prior and similiar study[26], it was shown that on a weekly basis CCH registered and treated as many or more patients as 13 other city hospitals combined. Secondly, virtually all of the patients registered at CCH were residents of Chicago and lastly, the same registrants used CCH as a primary care facility. Total visits at both hospitals combined ranged from 500 to 800 patients daily.

UIH was chosen as an alternative collection center. A doctor's strike at Cook County Hospital prompted most of the patients using the CCH facility to be routed to UIH. To be assured of the continued availability of all necessary patient data, the monitoring process continued at both hospitals.

Within the hospital, data was collected from three areas; the Emergency Room, Admissions*, and Pediatrics. Information was sought from these clinics because the majority of the study subjects were non-appointment patients only who sought medical attention for acute health disorders. It was felt that the majority of persons suffering from acute health effects of air pollution would dispense with the regular scheduling of a formal appointment and seek immediate medical attention. Another equally significant reason is that data obtained from hospital records in these areas was considered reliable and valid as a doctor's diagnosis accompanied each registered patient who had been treated. Data forms were discarded for any patient who left without a diagnosis, even if the complaint was acceptable to the study.

The Chicago Lung Association was initially subcontracted to acquire the necessary patient information and to work with project personnel in the

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*The admission of patients into UIH was not the sole function of the UIH Admission Clinics. Staff physicians also handled scheduled appointments for checkup, rechecks, etc. and treated patients with unscheduled appointments.

development of a collection system. A collection sheet entitled "Hospital Record of Patients with Respiratory and Cardiac Conditions" (hereafter referred to as the data form) was drafted.

After a month of collection, the arrangement with the Chicago Lung Association was found to be unneeded and the contract was terminated. A morbidity coordinator was added to the research team to supervise the data collection procedures and the subsequent processing of data. Clerical personnel were added to the project and trained to collect data from each hospital thrice weekly.

Data collection officially began April 12, 1977, and spanned one year, terminating on April 14, 1978. The morbidity collection team gathered data for every Tuesday, Wednesday and Thursday of each week from both hospitals. By omitting collection of data for weekends, days surrounding weekends (Fridays and Mondays), and holidays, a fairly regular pattern of visitation (in regards to type and number of patients) was established.

Data was collected at each hospital site by a 2 member team. Data forms were filled out to record information for each person whose illness was applicable to the study. Patient information transcribed on each form consisted of data and time of visit, address, age, race, sex, complaint, physician's diagnosis and whether patient was or was not a hospital admission. The patient's name was only written on the form if this was the only means of defining the sex.

Immediately after the data had been collected, the team validated their work by switching hospital and data forms. In such a manner they challenged each other's work and corrected any mistakes or irregularities. Validation completed the on-site hospital collection procedure.

After data had been collected, number codes were assigned for community area, census tract, patient's complaints and physician's diagnosis. Each form was then stamped with a six-digit sequence/reference number, codes were checked and sent out to be keypunched. When the keypunched cards and computer lists were returned, they were verified against the original data form for possible error. The data forms, cards, and lists were filed in sequential order for later analysis.

The only patient records utilized from CCH Pediatrics were log sheets on which general information was listed as a registration process. The

sex of the patient and the physician's diagnoses were not specified and formal patient records, which would have included these two items, could not be made accessable to the collection team. In light of the unavailability of complete and valid patient information, CCH Pediatrics was terminated as a source of data in December, 1977. All data collected to this point was deleted from the study. CCH Emergency Room and Admissions account for all CCH patients included in this study whose age was 15 years or older.

Two major groups of diseases and illnesses that could be considered as probable health consequences of existing air pollution levels were first compiled by key project personnel prior to the start of official data collections. All of these health disorders (listed in Table 1) were medically diagnosable, and of a respiratory or cardiovascular nature. Numerous past studies on air pollution versus health have shown that most highly urbanized populations experience a constant barrage of assorted ambient pollutants and as a result will suffer more severly and more frequently from these two categories of illnesses than from any other major disease grouping(s).

A code number was assigned to each diagnosis. This number served as a general indicator of type (respiratory or cardiovascular) and severity (acute or chronic) of the diagnosis. This number system also proved to be a useful training and reference guide for hospital data collectors. After computing daily averages of patients for eight disease groups (Table 2), three groups were excluded from the final analysis because of too few cases to obtain stable statistical measurements. As a result, the following disease groups were included in the final regression models:

1. All respiratory diagnoses (Group 8)-TRD
2. Allergic conditions and upper respiratory infections (Group 4)-RD1
3. Acute bronchial and lower respiratory infections (Group 5)-RD2
4. All cardiac diagnoses (Group 3)-TCD
5. Hypertension and vascular heart disease (Group 2)-CD1

Climatological Data

The U.S. Department of Commerce, National Oceanic and Atmospheric Administration (NOAA) maintains a local climatological data bank comprising Chicago-area stations. The data bank currently monitors meteorological variables at 3 stations within the city of Chicago; on the University of

TABLE 1

LIST OF CARDIAC AND RESPIRATORY DIAGNOSES

Group 1. Organic, Arteriosclerotic and Heart Failure

Diagnostic code No.	Diagnosis
02	Angina
02	Angina Pectoris
02	Impending Myocardial Infarction
02	Unstable Angina
03	Cardiac Arrhythmias
03	Arrhythmias
03	Heart Flutter
03	Atrial Fibrillation
03	Bradycardia
03	Fibrillation
03	Flutter
03	Premature Ventricular Constriction
03	Tachycardia
03	Ventricular Fibrillation
03	W.P.W. Syndrome
10	Cardiac Arrest
10	Cardiac Decompensation
10	C.H.F.
10	Possible Heart Failure
10	Pulmonary Edema
10	Ventricular Failure
11	Ischemic Heart Disease
11	Myocardial Ischemia
19	Coronary Occlusion
19	Myocardial Infarction/Occlusion
21	Palpitations
21	Abnormal EKG
21	Cardiomyopathy
21	Congential Heart Disease
21	Other Cardiac Conditions
22	Pericarditis
22	Bacterial Pericarditis
22	Viral Pericarditis
36	Aortic Incompetence (Insufficiency)
36	Aortic Stenosis
36	Chronic Rheumatic Heart Disease
36	Click
36	Click Syndrome
36	Systolic Click
36	Valvular Pathology
36	Valvular Syndrome
36	Mitral Regurgitation
36	Mitral Stenosis
36	Mitral Valve
36	Murmur
36	Rheumatic Heart Disease
40	ASHD/CAD
43	A.V. Block
44	Coronary Pulmonale
46	Endocarditis
47	Coronary Heart Disease
52	Unspecified Cardiac Problems

Group 2 Hypertensive and Vascular Heart Disease

Diagnostic code No.	Diagnosis
15	Hypertension
15	Hypertensive Heart Disease
33	Dizziness/Vertigo
33	Fainting
33	Vasovagal Attack
33	Syncope
37	Vascular Disease
37	Vascular Insufficiency
50	Cerebral Vascular Attack
50	Stroke

Group 3. Total Cardiac Diagnoses - In Alphabetical Order

1-21 Abnormal EKG
1-02 Angina
1-02 Angina Pectoris
1-36 Aortic Incompetence (Insuff.)
1-36 Aortic Stenosis
1-03 Arrhythmias
1-43 ASHD/CAD
1-43 A.V. Block
1-03 Atrial Fibrillation
1-22 Bacterial Pericarditis
1-03 Bradycardia
1-10 Cardiac Arrest
1-03 Cardiac Arrhytimia
1-10 Cardiac Decompensation
1-21 Cardiomyopathy
2-50 Cerebral Vascular Attack
1-10 C.H.F.
1-36 Chronic Rheumatic Heart Disease
1-36 Click
1-36 Click Syndrome
1-21 Congential Heart Disease
1-47 Coronary Heart Disease
1-19 Coronary Occlusion
1-44 Coronary Pulmonale
2-33 Dizziness/Vertigo
1-46 Endocarditis
2-33 Fainting
1-03 Fibrillation
1-03 Flutter
1-03 Heart Flutter
2-15 Hypertension
2-15 Hypertensive Heart Disease
1-02 Impending Myocardial Infarction
1-11 Ischemic Heart Disease
1-36 Mitral Regurgitation
1-36 Mitral Stenosis
1-36 Mitral Valve
1-36 Murmur
1-19 Myocardial Infarction/Occlusion
1-11 Myocardial Ischemia
1-21 Other Cardiac Conditions
1-21 Palpitations
1-22 Pericarditis
1-10 Possible Heart Failure
1-03 Premature Ventricular Contractions (P.V.C.)
1-10 Pulmonary Edema
1-36 Rheumatic Heart Disease
2-50 Stroke
2-33 Syncope
1-36 Systolic Click
1-03 Tachycardia
1-52 Unspecified Cardiac Problems
1-02 Unstable Angina
1-36 Valvular Pathology
1-36 Valvular Syndrome
2-37 Vascular Disease
2-37 Vascular Insufficiency
2-33 Vasovagal Attack
1-10 Ventricular Failure
1-03 Ventricular Fibrillation
1-22 Viral Pericarditis
1-03 W.P.W. Syndrome

Group 4. Allergic Conditions and Acute Respiratory Infections.

Diagnostic Code No.	Diagnosis	Diagnostic Code No.	Diagnosis
01	Allergic Cold	34	Tonsillitis
01	Allergic Rhinitis	34	Follicular Tonsillitis
01	Hayfever	34	Hypertrophic Tonsillitis
17	Laryngitis	35	Cold
17	Pharyngitis	35	Coryza
17	Sore Throat	35	Cough
30	Paranasal Sinusitis	35	Upper Respiratory Infection (URI)
30	Sinusitis	42	Allergy (Croup)
32	Strep Throat		

Group 5. Acute Bronchial and Lower Respiratory Infections

Diagnostic Code No	Diagnosis	Diagnostic Code No.	Diagnosis
04	Asthma	23	Pleurisy
04	Bronchial Asthma	23	Pleuritis
06	Bronchitis	26	Bronchiolitis
06	Acute Bronchitis	26	Pneumonia
06	Tracheobronchitis		

Group 6. Other Related Respiratory Disorders

Diagnostic Code No.	Diagnosis	Diagnostic Code No.	Diagnosis
08	Chest Pain	25	Respiratory Distress (Failure)
03	Atypical Chest Pain	27	Respiratory Alkalosis
08	Musculoskeletal Chest Pain	27	Hyperventilation Syndrome
08	Unspecified Chest Pain	31	Dypsnea (Shortness of Breath)
25	Pulmonary Insufficiency		

RESPIRATORY DIAGNOSIS CON'T

Group 7. Chronic Restrictive Respiratory Infections and other Pulmonary Conditions

Diagnostic Code No	Diagnosis	Diagnostic Code No.	Diagnosis
09	COPD	28	Lung Abscess
09	Emphysema	28	Pulmonary Congestion
09	Restrictive Lung Pattern	28	Purulent Congestion
13	Granulomatous Disease	28	Respiratory Tract Infection
13	Pneumoconioses	28	Viral Syndrome
13	Sarcoidosis	39	Pleural Effusion
13	Tuberculosis (TB)	51	Unknown Pulmonary Problem

Group 8. Total Respiratory Diagnoses - In Alphabetical Order

5-06	Acute Bronchitis
4-01	Allergic Cold
4-01	Allergic Rhinitis
4-42	Allergy (Croup)
5-04	Asthma
6-08	Atypical Chest Pain
5-26	Broncheolitis
5-04	Bronchial Asthma
5-06	Bronchitis
6-08	Chest Pain
4-35	Cold
7-09	COPD
4-35	Coryza
4-35	Cough
6-31	Dypsnea (Shortness of Breath)
7-09	Emphysema
4-34	Follicular Tonsillitis
7-13	Granulomatous Disease
4-01	Hayfever
4-34	Hypertrophic Tonsillitis
6-27	Hyperventilation Syndrome
4-17	Laryngitis
7-28	Lung Abscess
6-08	Musculoskeletal Chest Pain
4-30	Paranasal Sinusitis
4-17	Pharyngitis
7-39	Pleural Effusion
5-23	Pleurisy
5-23	Pleuritis
7-13	Pneumoconioses
5-26	Pneumonia
7-28	Pulmonary Congestion
6-25	Pulmonary Insufficiency
7-28	Purulent Congestion
6-27	Respiratory Alkalosis
6-25	Respiratory Distress (Failure)
7-28	Respiratory Tract Infection
7-09	Restrictive Lung Pattern
7-13	Sarcoidosis
4-30	Sinusitis
4-17	Sore Throat
4-32	Strep Throat
4-34	Tonsillitis
5-06	Tracheobronchitis
7-13	Tuberculosis (TB)
7-51	Unknown Pulmonary Problems
6-08	Unspecified Chest Pain
4-35	Upper Respiratory Infection
7-28	Viral Syndrome

Table 2

Daily Means of Emergency Room Visits for Major Disease Groups

	Study Phase*			
	I	II	III	IV
All respiratory diagnoses (Group 8)	83.15	88.38	90.77	66.64
Allergic conditions and upper respiratory infections (Group 4)	55.15	58.74	61.03	45.14
Acute bronchial and lower respiratory infections (Group 5)	16.70	16.67	16.41	13.94
Other related respiratory disorders (Group 6)	7.15	8.07	7.96	4.72
Chronic restrictive respiratory infections and other pulmonary conditions (Group 7)	4.15	4.91	5.37	2.83
All cardiac diagnoses (Group 3)	20.25	21.33	21.51	16.03
Organic, arteriosclerotic and heart failure (Group 1)	4.75	5.02	5.22	4.25
Hypertensive and vascular heart disease (Group 2)	15.50	16.31	16.29	11.78
Number of observations (days)	20	42	131	36

Note: *See Multiple Regression Analysis in Section 5 in regard to four study phases.

Chicago campus; at Midway Airport; and near Buckingham Fountain in the loop. However, the Midway Airport site presented the most concise set of daily data as maintained by a complete and precision monitoring system. For this purpose, daily climatological variables in the model are represented by Midway Airport (as published by U.S. Department of Commerce, NOAA).

The daily measurements used in the analysis were average temperature in degrees fahrenheit, total water precepitation in inches, average wind speed in miles per hour, average relative humidity in per cent, hours of possible sunshine in per cent, and sky cover in tenths.

Air Pollution Data

The city of Chicago maintains a large and complex air pollutant monitoring network. A total of 34 sites have been created and maintained over the past fourteen years. Although each site is within the boundaries of the city, the governing responsibility of each site might lie with any one of four agencies (the Federal Environmental Protection Agency, the State of Illinois EPA, the City of Chicago Department of Environmental Control (DEC) and the U.S. Public Health Services). Name, location and site-codes for each station may be found in Figure 1. It might be noted that although all sites are registered on the chart, over a period of time, there have been stations shut-downs, full closures and alternations in the equipment utilized. However, this is the most complete and concise listing of what sources are available in metropolitan Chicago.

A variety of pollutant parameters are measured within the network, including suspended particulate (total, benzene soluble, soiling index, etc.), and elements as tin, nickel, lead, chromium, barium and arsenic, and gases such as carbon monoxide, sulfur dioxide, nitric oxide, nitrogen dioxide, total oxidants, ozone, methane and hydrocarbons. As expected, a variety of collection and analysis methods are utilized in each process, offering an overlap of data files. It is quite possible for more than one agency to maintain surveillance of these air contaminants at one site. Albeit confusing, these data analysis results should be more meaningful in tandem with concurrent results from several monitors operated by independent agencies.

As previously mentioned, most of the city's air quality data is recorded

Figure 1. Location of air monitoring sites and 76 community areas in Chicago

by the Department of Environmental Control. The DEC maintains two networks: (1) the 26 station Chicago Air Sampling Network (CASN) and (2) 8 station Telemetric Air Monitoring Network (TAMN). Monitored by the Technical Services Division, the air contaminants currently monitored are sulfur dioxide, particulate, carbon monoxide, nitrogen dioxide, nitric oxide and ozone. The CASN network currently monitors 3 air contaminants; SO_2 concentrations measured by West-Gaeke method on a 24 hour sample every six days; TSP concentrations using a hi-vol sampler for 24 hours every three days; and NO_2 concentrations using a modified christie method every six days.

The TSMN network measures the remaining air contaminants on a daily basis, tabulating 96 values per day (reported on a 15-minute basis): again SO_2, utilizing a conductivity analyzer every 15 minutes, every day; CO concentrations measured with a non-dispersive infarred analyzer and NO and NO_2 (again) concentrations using the colorimetric method analysis.

Supplementary data was later provided by one site, Polk Street, at the University of Illinois Medical Center monitoring all above noted pollutants, plus ozone. However, this site is monitored by the State of Illinois Environmental Protection Agency on a daily basis.

The Chicago Department of Environmental Control (DEC) was petitioned for all Chicago air contaminant data monitored from the time period April 1, 1977 through April 30, 1978. This utilized both the 26 station CASN network and the 8-station TAMN network. Data for the TAMN stations was marked on monthly log sheets, having been recorded hourly for CO, O_3, NO_2 and SO_2. CASN stations were recorded in the data format published by the DEC. An example is displayed in Table 3. Unfortunately there was to be little uniformity of collection periods; TSP was monitored every 3 days, while SO_2 and NO_2 were monitored every six days (the same day). Of course, all TAMN stations monitor the environment on a daily basis. Due to this infrequency of pollutant monitoring, of 150 study days for which emergency room visit records were collected, SO_2 and NO_2 were measured on 20 of these days, and TSP on 42 of the days.

Separate master files were created for each pollutant, TSP, SO_2 and NO_2 from the CASN network, accordingly for each day they were monitored. However, after examining more air data for CO, O_3 and SO_2 (TAMN data) a decision was made to eliminate these pollutant files for a variety of

Table 3. CITY OF CHICAGO, DEPARTMENT OF ENVIRONMENTAL CONTROL

CHICAGO AIR SAMPLING NETWORK

Sulfur Dioxide SO_2 PPM April, 1977

DATE		1	7	13	19	25	NO.
A - 03	Taft	.024	.007	.009	.015	.008	5
B - 04	Lakeview	.014	.003	.022	.012	.010	5
C - 11	Steinmetz	.039	.020	.013	.014	.014	5
D - 12	Cooley	.020	.062	.022	.017	.010	5
F - 05	GSA	.023	.026	.028	.019	.010	5
H - 06	Austin	.018	.016	.015	.019	.008	5
I - 14	Farr	-	-	-	-	-	-
J - 15	Kelly	.022	.021	.019	.008	.010	5
K - 07	Lindblom	.023	.018	.014	.007	.011	5
M - 09	Stevenson	.018	.017	.014	.007	.011	5
N - 16	Calumet	.013	.011	.011	.006	.007	5
O - 17	CVS	.051	.018	.010	.006	.013	5
P - 10	Fenger	.021	.018	.010	.006	.013	5
Q - 18	Carver	.022	.024	.010	.011	.012	5
R - 19	Clay	M	M	M	M	M	M
S - 25	Kenwood	.019	.021	.009	.025	.013	5
T - 20	Sullivan	.020	.028	.021	.017	.008	5
V - 28	Logan Sq.	-	-	-	-	-	-
W - 21	Hale	.015	.024	.043	.020	.006	5
AVERAGE		.023	.021	.017	.014	.010	80
U - 29	Von Steuben	-	-	-	-	-	-
YY- 22	Washington	.019	.017	.027	.007	.012	5
E - 32	S.W.F.P.	.010	.011	.008	.006	.005	5
OO- 30	Anthony	.009	.009	.040	.025	.006	5
Y - 31	Adams	.031	.014	.030	.039	.018	5
34	CRIB	.011	.013	K	K	K	2
37	Edgewater	.016	.020	.017	.013	.011	5
AVERAGE		.021	.019	.109	.015	.010	107

reasons. This design would have been ideal to study, allowing us to use nearly all the hospital data (TAMN data is daily data, versus 20 days usage with solely the CASN network). However, the TAMN data could not be used because of the disproportionately large amount of station shut-downs within the study frame. There were significant periods of time during the study period in which only 2-3 out of the 8 stations were open (for one parameter). In addition, TAMN data was often subject to a careless rounding-off process onto the log sheet. For example, all measurements for SO_2 were reported to one significant digit, whereas most stations will show a difference in SO_2 levels only when at least two significant digits are reported.

In order to supplement the CASN station data, data under the auspices of the State of Illinois Environmental Protection Agency was added to the statistical model. This site, Polk Street, at the University of Illinois Medical Center was chosen since it was the closest to the Cook County - University of Illinois Medical Center complex. Polk Street maintains daily files of SO_2, CO, NO, NO_2 and O_3 and was not plagued by shut-downs or alterations in management. Therefore, again separate files, by date, were created for each pollutant.

After all air contaminant files were transcribed, keyed on-line and verified, a process was initiated to create community area exposure levels.

This technique was appropriate with CASN data only however, since readings were obtained from only one station in the city, community areas exposure levels could not be used for the Polk Street data. Consequently, the assumption was made that this one reading was representative of the entire city.

ESTIMATION OF MISSING POLLUTION MEASUREMENTS

The major problem faced with utilizing measurements from the city air pollution network was the fairly large amount of missing data. As shown in Table 4, out of 26 stations measuring TSP, 4 were closed down more than 25% of the time and therefore completely eliminated from the study. They were Farr (14), Clay (19), Logan Square (28) and Crib (34). Similarly, 5 out of 26 sites measuring SO_2 and NO_2 were eliminated (Farr, Clay, Logan Square, Von Steuben (29) and Crib).

For the remaining stations, the percentage of days shut-down ranged

TABLE 4

No. of Days Monitoring Station Shutdown

Station	TSP	SO_2	NO_2
Camp - 2	---	---	8
Taft - 3	23	4	4
Lakeview - 4	11	0	0
GSA - 5	8	5	5
Austin - 6	23	5	5
Lindblom - 7	22	3	3
Stevenson - 9	15	4	4
Fenger - 10	16	1	1
Steinmetz - 11	11	2	2
Cooley - 12	12	0	0
Farr - 14	128*	66*	66*
Kelly - 15	16	2	2
Calumet - 16	13	0	0
CVS - 17	17	1	1
Carver - 18	24	2	2
Clay - 19	132*	66*	66*
Sullivan - 20	32	3	3
Hale - 21	15	0	0
Washington - 22	17	3	3
Kenwood - 25	16	0	0
Logan Square - 28	73*	66*	66*
Von Steuben - 29	11	66*	66*
Anthony - 30	9	8	8
Adams - 31	15	2	2
SWFP - 32	24	1	1
Crib - 34	70*	30*	31*
Edgewater - 37	20	3	3
Total Days Measured	132	66	66

* Stations Eliminated from Study

from 0 to 25% for the duration of the study. (See Table 4). Since for these sites the amount of missing observations was fairly low, the data was supplemented with estimates of SO_2, NO_2 and TSP levels.

This estimation was done by multiple regression methods. For each site, the four closest sites were identified. Then, using monitoring days on which all 5 sites were open,a predictive equation was developed in which levels at the 4 surrounding sites were used as predictors. This was done for each site and each pollutant, since there was no evidence that a prediction equation at one site would be the same for all pollutants. In a number of instances, so many sites were closed down on a given day including a predictor station that alternate regression equations were developed and used in the manner described above. Tables 5,6,7 and 8 list the regression equations used and their R^2 values.

CALCULATION OF COMMUNITY AREA EXPOSURE LEVELS

To determine if there is a relationship between exposure to air pollution and morbidity, a method to measure exposure must be established. Obviously, to get the most accurate measure of a person's total exposure to air pollution on a certain day, the person's location throughout the day must be known and also the corresponding air pollutant levels at each location. This was not feasible utilizing the available resources.

However, the residential address of emergency room patients was available. Under the assumption that the average person spends a majority of his time in or near his home, the community area in which the patient resides was designated as his location. One major limitation to this process is that a number of people who travel long distances to work spend a large portion of their day away from home. Associated with daily absence from residence is the consideration of exposure to pollutants caused by a person's occupation. In the first case, it was noticed that most people do not work more than 12 hours a day, and so their residence would be most representative of their location for the whole day. In the second case, no means were available of obtaining the patient's occupation and work address.

By using measurements from Chicago's 26 air quality monitoring stations, a procedure was developed to estimate the level of pollutants in each community area. To accomplish this, the following criteria were

TABLE 5
PREDICATION EQUATIONS FOR TSP MISSING DATA

R^2	
0.30	Taft =0.908 x Von Steuben + 22.306
0.72	Lakeview =0.427 x Edgewater +0.396 x Sullivan +0.343 x Steinmetz -5.128
0.58	GSA =0.347 x Cooley +0.262 x Kenwood +28.692
0.58	Austin = 0.653 x Steinmetz +0.225 x Colley +19.39
0.73	Lindblom =0.548 x Calumet +0.258 x Kenwood +0.131 x Kelly +7.479
0.34	Stevenson =0.484 x Fenger +0.344 x Calumet +13.821
0.84	Fenger = 0.483 x Carver +0.336 x Stevenson +0.145 x CVS -5.15
0.74	Steinmetz =0.463 x Von Steuben +0.310 x Austin +9.631
0.55	Colley =0.373 x Lakeview +0.450 x GSA +0.395 x Edgewater +8.189
0.30	Kelly =0.756 x Lindblom +24.389
0.81	Calumet =0.784 x Lindblom +0.208 x CVS -0.070
0.81	CVS =0.476 x Anthony +0.241 x Calumet +0.108 x Adams +0.259
0.70	Carver =0.501 x Anthony +0.465 x Fenger + 12.665
0.62	Sullivan =0.329 x Lakeview +0.339 x Steinmetz +0.156 x Edgewater +3.593
0.57	Hale =0.906 x Calumet +23.222
0.80	Washington =0.787 x Carver +110.32
0.68	Kenwood =0.547 x Calumet +0.262 x SWFP +5.445
0.64	Von Steuben =0.269 x Lakeview +0.367 x Edgewater +0.149 x Taft +8.675
0.80	Anthony =9.773 x CVS +0.344 x Fenger +9.190
0.52	Adams =0.573 x CVS =0.434 x Fenger +41.445
0.54	SWFP =1.148 x Kenwood +4.645
0.65	Edgewater =0.462 x Lakeview +0.339 x Von Steuben +16.031

TABLE 6
ALTERNATE EQUATIONS FOR TSP MISSING DATA

R^2	
0.64	Lakeview =0.510 x Steinmetz +0.202 x Cooley +0.187 x Taft -0.684
0.63	Lindblom =0.327 x Kenwood +0.232 x Kelly +0.177 x Hale +0.108 x Stevenson +10.968
0.32	Stevenson =0.621 x Calumet +0.193 x Hale +13.261
0.58	Calumet =0.346 x Fenger +0.263 x CVS +0.208 x Kelly +11.596
0.77	Von Steuben =0.540 x Steinmetz +0.175 x Cooley +0.154 x Taft -1.621
0.53	Fenger =0.454 x Calumet +0.311 x CVS +0.042 x Carver +12.259
0.58	Lakeview =0.884 x Edgewater +9.1767
0.51	Austin =0.893 x Steinmetz +25.83
0.72	Lindblom =0.619 x Calumet +0.280 x Kenwood +11.937
0.67	Lindblom =0.859 x Calumet +12.253
0.82	Fenger =0.461 x Carver +0.483 x Stevenson -1.897
0.66	Steinmetz =0.738 x Von Steuben +18.904
0.52	Cooley =0.555 x Lakeview +0.552 x GSA +15.700
0.47	Cooley = 0.976 Lakeview +30.683
0.78	CVS = 0.531 x Anthony +0.244 x Calumet +7.253
0.74	CVS =0.683 x Anthony +12.155
0.64	Carver =0.847 x Anthony +12.100
0.53	Von Steuben =0.620 x Lakeview +22.282

TABLE 7
PREDICTION EQUATIONS FOR SO_2 MISSING DATA

R^2	
0.51	Taft =0.339 x Steinmetz +0.209 x Edgewood +0.001
0.64	Lakeview =0.478 x Edgewater +0.253 x Steinmetz +0.001
0.22	GSA =0.790 x Kelly +0.784 x SWFP +0.005
0.51	Austin =0.415 x Steinmetz +0.194 x Cooley +0.147 x Hale +0.001
0.65	Lindblom =0.517 x Kelly +0.140 x Kenwood +0.301 x Stevenson
0.71	Stevenson =0.862 x Calumet +0.243 x Lindblom +0.006 x Hale
0.66	Fenger =0.191 x CVS +0.176 x Stevenson +0.343 x Calumet +0.127 x Carver
0.61	Steinmetz =0.588 x Lakeview +0.656 x Taft
0.66	Cooley =1.436 x Edgewater -0.810 x Lakeview +0.335 x Austin +0.001
0.58	Kelly =0.623 x Lindblom +0.071 x Hale +0.001
0.72	Calumet =0.360 x Stevenson +0.976 x CVS +0.124 x Fenger
0.50	CVS =1.298 x Calumet +0.245 x Adams +0.001
0.47	Carver =0.714 x Fenger +0.147 x Adams +0.001
0.65	Sullivan =0.544 x Edgewater +0.339 x Steinmetz +0.001
0.30	Hale=0.703 x Stevenson +0.828 x Calumet +0.004
0.32	Washington =0.334 x Adams +0.244 x Anthony +0.003
0.26	Kenwood =0.726 x Lindblom +0.007
0.28	Anthony =0.273 x Adams +0.243 x Washington +0.001
0.47	Adams =0.452 x CVS +0.355 x Washington +0.002
0.23	SWFP =0.258 x Lindblom +0.100 x GSA +0.002
0.70	Edgewater = 0.432 x Lakeview +0.372 x Sullivan +0.250 x Taft

TABLE 8

PREDICTION EQUATIONS FOR NO_2 DATA

R^2	
0.27	Camp =1.103 x Cooley +0.004
0.38	Taft =0.676 x Lakeview +0.005
0.70	Lakeview =0.351 x Steimetz +0.249 x Taft +0.267 x Sullivan +0.176 x Edgewater +0.006
0.29	GSA =0.687 x Cooley +0.286 x SWFP +0.008
0.35	Austin =0.488 x Steimetz +0.469 x Cooley +0.006
0.52	Lindblom =0.480 x Kelly +0.430 x Calumet +0.003
0.14	Stevenson =0.394 x Hale +0.018
0.45	Fenger 0.287 x Calumet +0.227 x CVS -0.074 x Carver +0.120 x Stevenson +0.005
0.57	Steinmetz =0.587 x Lakeview +0.181 x Austin +0.004
0.66	Cooley =0.422 x Lakeview +0.157 x GSA +0.100 x Austin +0.012
0.57	Kelly =0.510 x Hale +0403 x Lindblom +0.006
0.52	Calumet =0.296 x Lindblom +0.254 x Fenger +0.193 x Stevenson +0.200 x CVS +0.007
0.35	CVS =0.355 x Calumet +0.273 x Adams +0.160 x Anthony +0.008
0.09	Carver =0.485 x Anthony -0.653 x Washington +0.285 x Adams +0.030
0.46	Sullivan =0.477 x Lakeview +0.263 x Steinmetz
0.65	Hale =0.473 x Calumet +0.315 x Kelly +0.152 x Stevenson +0.003
0.40	Washington =0.339 x Anthony +0.300 x Adams -0.093 x Carver +0.016
0.41	Kenwood =0.612 x SWFP +0.242 x Kelly +0.010
0.30	Anthony =0.556 x Washington +0.286 x CVS +0.003
0.33	Adams =0.390 x CVS +0.413 x Washington +0.007
0.50	SWFP =0.445 x Kenwood +0.211 x GSA +0.209 x Crib -0.166 x Kelly +0.006
0.32	Edgewater =0.399 x Lakeview +0.243 x Steinmetz +0.013

ALTERNATE EQUATIONS FOR NO_2 MISSING DATA

R^2	
0.28	Washington =0.288 x Adams +0.427 x Fenger +0.013
0.21	Anthony =0.586 x Fenger +0.248 x CVS +0.008

used:

1. If a station existed in the community area, that station's measurements were used for the community area.
2. If two or more stations were located in a community area, their daily measurements were averaged.
3. If no station existed in the community area, the closest stations were located and their distances to the center of the community area were estimated. The community area daily measurements were calculated as the weighted average (inversely proportionate to the distance of each site from the CA) of all the surrounding sites.

Thus, for each of the three pollutants the exposure levels of 76 community areas were generated for each day by a Fortran IV program.

CALCULATION OF DISEASE SPECIFIC AVERAGE EXPOSURE LEVELS

Once a set of community area exposure levels was completed for each of the three pollutants (TSP, SO_2 and NO_2) the next step was to obtain personal exposure levels for each of the 21,000 patients' records. This was done by identifying the date on which the patient visited the emergency room and the community area in which the subject resided. The community exposure levels for that day then were added to the patient record.

The exposure levels of all patients in each specific disease category were combined for each pollutant. Then an average exposure level was obtained by dividing the sum of the individual exposure levels by the number of patients in the disease category. If each patient's exposure level is X_i and the number of patients in any specific disease category is n, an average exposure level (AEL) to a specific pollutant for that disease category can be expressed as follows:

$$AEL = \frac{\sum_{i=1}^{n} X_i}{n} \qquad (i)$$

MULTIPLE REGRESSION ANALYSIS

Multiple regression techniques were used to examine if any specific kinds of air pollutants have significant impact on the variation of emergency room visits for cardiac and respiratory conditions, even after

controlling for daily climatologic changes and days-of-week effects.

The dependent variable was the percentage of excess visits for cardiac or respiratory disease, which was calculated as follows:

$$\% \text{ of excess visits} = \frac{100\ (X_i - \bar{X})}{\bar{X}} + 100 \qquad \text{(ii)}$$

where X_i is the number of emergency room visits for a specific disease on the i-th day and $\bar{X}$ is the average number of visits for that disease during the study period. To avoid a negative sign on per cent of excess visits, 100 was added to the right side of the equation. If X_i is greater than the average, % of excess visits becomes greater than 100%. If X_i is smaller than the average, its percentage becomes less than 100%.

The justifications to use per cent of excess visits as a morbidity index are as follows: (1) a majority of people who utilize both Cook County Hospital and University of Illinois Hospital are persons from low-income families whose health condition may be more affected by air pollution than others, and (2) it is assumed that per cent of excess visits would not be influenced by the size of the risk population (although unknown) because the risk population would not be changed during the one-year study period unless there were great changes in policies and administrations for patient care services in these two hospitals. Frequency distributions of patients in both hospitals are shown according to race, sex, age and diagnostic group in Tables 23-26 in the Appendix.

Three types of independent variables were used; (1) air pollutants, (2) climatological measurements and (3) days of the week as dummy variables. As described in Air Pollution Data, the number of observations was different from one pollutant to the others, so that only one pollutant was included in each equation. Accordingly, four different data sets were prepared and regression analysis was done in four phases as follows:

Phase I. Only these days on which SO_2 and NO_2 were monitored by the CASN were used (i.e., 20 study days). Two models were developed:

SO_2-I (based on average exposure levels of patients)

NO_2-I (based on average exposure levels of patients)

Phase II. Only those days on which TSP was monitored by the CASN were used (i.e., 42 study days). Only one model was developed:

TSP-II (based on average exposure levels of patients)

Phase III. Only those days on which pollution measurements from the University of Illinois Medical Center at Polk Street were recorded were used (i.e., 131 study days). Four models for each disease group were developed:

SO_2-III (based on daily averages at Polk Street).

NO_2-III (based on daily averages at Polk Street).

CO -III (based on daily averages at Polk Street).

NO -III (based on daily averages at Polk Street).

Phase IV. A separate analysis of ozone in each disease group was done by including all study days between May 17, 1977 and August 31, 1977, the period when daily maximum temperature was usually higher than 75° F. This allowed 36 study days to be used. Only one model for each disease group was developed:

O_3-IV (based on daily averages at Polk Street).

Daily climatological measurements used for analysis were (1) average temperature in degrees fahrenheit, (2) total water precipitation in inches, (3) wind speed in miles per hour, (4) humidity in per cent, (5) possible sunshine in per cent and (6) sky cover in tenths. Finally, days of the week (Tuesday, Wednesday and Thursday) were included in all models as dummy variables because frequency of hospital visits may be higher on a specific day of the week than on other days.

The general model to be tested in this study is expressed as follows:

$$Y=\beta_0 X_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_3 + \beta_4 X_4 + \ldots + \beta n Xn + \varepsilon \qquad \text{(iii)}$$

where X_0= 1 for all observations, and where X_1 and X_2 are used for day-of-week identification as follows:

X_1	X_2	
1	0	= Tuesday
0	1	= Wednesday
0	0	= Thursday

and where β_0, β_1, ..., βn = regression coefficients (β_0 is constant since

since $X_0 = 1$)

X_3 = a specific pollutant

$X_4, \ldots, X_n$ = climatological variables included in the equation

$n=3+m$ (m is the number of climatological variables added to the equation.)

ε = random error term, and

Y = % of excess visits in a specific disease group.

In computation procedures, a specific pollutant was introduced in each regression analysis as a first step, then two dummy variables (Tuesday and Wednesday) as a second step, and climatological variables according to the stepwise method. Since our interest was centered on the air pollution variable, only climatological variables which had significant or meaningful influences on the regression model were included according to the criterion that the F value of the regression coefficient is greater than or close to a significant level ($p<.10$).

SECTION 6

RESULTS AND DISCUSSION

MODELS FOR TOTAL RESPIRATORY DIAGNOSES (TRD)

Eight regression models were created to examine air pollution effects on total respiratory diagnoses (TRD) as shown in Table 9. The proportion of the variation in percent of excess visits for TRD ranged from 19% in model 4 (NO_2-I) to 36% in model 2 (SO_2-I). According to the F ratios, the overall test for goodness of fit of the regression equation was statistically significant in models 1, 3, 5, 6, 7 and 8. All regression coefficients of the pollutants were not statistically significant except NO in model 7 (NO-III). Humidity and sunshine as climatological variables were not included in any of the models. Wind speed was included only in model 8 (O_3-IV). Daily average temperature had the most significant impact on TRD in models 1, 3, 5, 6 and 7. That is, as the temperature decreases, the number of patients with respiratory conditions increases. Only the last model for ozone had significant days-of-week effects of TRD.

By paying attention to each model, the strength of the relationship between a specific pollutant and TRD can be examined. Model 1 shows that the partial regression coefficient of TSP, .230, was greater than its standard error, .170, but was not significant, controlling for average temperature and precipitation which were meaningful additions to the model.

Models 2 and 3 examined SO_2 effects on TRD, although their SO_2 indices were different. The regression coefficient of SO_2 in model 2 was considerably greater than its standard error, but not statistically significant because the sample size was too small. The regression coefficient of SO_2 in model 3, on the other hand, indicated no association between SO_2 and TRD. SO_2 measurements at the University of Illinois Medical Center on Polk Street were used in model 3 as an alternative of the average exposure levels (AEL) of patients, since there were only 20 observations of SO_2 measurements from the Chicago Air Sampling Network (CASN). According to the correlation coefficient between SO_2 from the CASN and SO_2 from Polk Street, 0.477, as provided in Table 19 in the Appendix, SO_2 levels at Polk Street did not reflect well the average exposure levels of patients. Also, differences in the regression coefficient of SO_2 between models 2 and 3 can be attributed

TABLE 9 MORBIDITY MODELS: TOTAL RESPIRATORY DIAGNOSES (TRD)

		(1) TSP-II	(2) SO_2-I	(3) SO_2-III	(4) NO_2-I	(5) NO_2-III	(6) CO-III	(7) NO-III	(8) O_3-IV
Multiple R		.52	.60	.58	.44	.58	.57	.57	.59
R^2		.27	.36	.33	.19	.34	.33	.33	.35
F ratio		2.66**	1.55	10.26***	.88	10.44***	12.21***	15.35***	3.21**
d.f.		5,36	5,14	6,124	4,15	6,124	5,125	4,126	5,30
Constant		110.7	72.2	123.6	140.0	123.5	119.5	127.4	73.8
Pollutant	reg.	.230	2053	93	-638	80.4	3.39	116.8*	399.4
	s.e.	.170	1331	240	643	86.2	2.90	63.1	437.0
	F	(1.82)	(2.38)	(.15)	(.98)	(.87)	(1.37)	(3.43)	(.836)
Temperature	reg.	-.58***		-.63***		-.68***	-.65***	-.64***	
	F	(7.73)		(34.37)		(44.68)	(46.73)	(45.78)	
Wind speed	reg.								-2.72
	F								(2.09)
Humidity	reg.								
	F								
Precipitation	reg.	-14.78	-19.14	-9.11	-20.4	-9.20			
	F	(2.45)	(2.27)	(1.83)	(2.37)	(1.92)			
Sky cover	reg.		2.27	1.21*		1.26*	.995		4.17**
	F		(1.64)	(3.23)		(3.48)	(2.43)		(6.59)
Sunshine	reg.								
	F								
Days-of-week effects									
Tuesday	reg.	5.19	-10.96	.37	-16.62	.55	.82	.24	23.57**
	F	(.25)	(.41)	(.01)	(.59)	(.00)	(.02)	(.00)	(5.3)
Wednesday	reg.	1.93	11.24	4.66	-3.71	4.40	5.06	4.91	32.48***
	F	(.03)	(.45)	(.75)	(.04)	(.67)	(.89)	(.84)	(9.4)

Note: 1) For each independent variable, regression coefficient, standard error (for pollutant only) and F statistic are represented on lines of reg., s.e. and F, respectively.
2) The level of significance is marked as asterisk: *(α=.10), ** (α=.05), and *** (α=.01)

to the difference in correlation coefficients between TRD and SO_2 from the CASN (0.364) and between TRD and SO_2 from Polk Street (-0.035), as shown in Table 15 in the Appendix. Using air pollution measurements from Polk Street as an alternative of the AEL was probably the greatest limitation in this study, although only air pollution data from Polk Street were available at the time of data collection.

Association of NO_2 with TRD was not significant in both models 4 and 5. Most of the variation of TRD in model 5 (34%) was explained by average temperature, sky cover and precipitation. The correlation coefficient between NO_2 from the CASN and NO_2 from Polk Street was only 0.179 (Table 19 in the Appendix). Correlation coefficients between NO_2 and TRD were -0.172 based on the CASN data and 0.011 based on the data from Polk Street (Table 15 in the Appendix).

Association between CO and TRD was not significant, after partitioning effects of temperature and sky cover which were more strongly related to TRD than CO was. However, the standard error of CO was smaller than its regression coefficient.

Model 7 shows significant association between NO and TRD, controlling for average temperature and days-of-week effects. Other climatological variables were not included in the model because of no meaningful contributions to the increase in the variation or F ratio by adding them to the equation. In short, the regression coefficient of NO, 116.8, was significant at $p<.10$ ($3.43 > F(.10, 1, 120) = 2.75$). Thirty-three per cent of the variation in TRD was accounted for by average temperature, NO and days-of-week variables.

Model 8 indicated no significant association between O_3 and TRD. The F statistic of O_3 was only 0.836 which was far from the significant level, $F(.10, 1, 30) = 2.88$. Climatological variables included in the model were sky cover (significant at $P< .05$) and wind speed. F statistics of day-of-week variables were both statistically significant; $5.30 > F(.05, 1, 30) = 4.17$ on Tuesday and $9.40 > F(.01, 1, 30) = 7.56$ on Wednesday. This means that the number of emergency room visits for TRD varied significantly among the days of the week. No reason was found for this.

MODELS FOR ALLERGIC CONDITIONS AND UPPER RESPIRATORY INFECTIONS (RD1)

Table 10 is a summary of eight models for allergic conditions and upper respiratory infections (RD1). The variation of RD1 explained by the regression models ranged from 29% in models 1 and 4 to 40% in model 2. According to the F ratio used to test the overall goodness of fit of the regression equation, statistically significant levels were observed in models 1, 3, 5, 6, 7 and 8, but not in models 2 and 4 from phase I analysis which had only 20 observation days.

Out of eight models, only model 7 (NO-III) indicated a significant impact of the pollutant, or NO, on RD1. The standard error of NO, 85.4, was about half the size of its regression coefficient, 161.5. Its F statistic, 3.58, was greater than F (.10, 1, 120) = 2.75 but smaller than F (.05, 1, 120) = 3.92. Average temperature had the strongest association with RD1 in which the F statistic of its coefficient was 31.88 > F (.01, 1, 120) = 6.85. Precipitation, sky cover and wind speed were included in the model because of their considerable influences on both the dependent and other independent variables. The days-of-week variables were not significant at all as shown in their F statistics, 0.16 and 0.46. Thirty-four per cent of the variation of RD1 was explained by the linear model including NO, average temperature, wind speed, precipitation, sky cover and the days-of-week variables, with F ratio, 9.25> F (.01, 1, 120) = 6.85.

Among seven other models, coefficients of SO_2 in both models 2 and 3 were considerably greater than their standard errors, although their F values did not reach a significant level. There is little reason to support the strong association between RD1 and other pollutants, such as TSP, NO_2, CO, and O_3 according to models 1, 4, 5, 6 and 8.

MODELS FOR ACUTE BRONCHIAL AND LOWER RESPIRATORY INFECTIONS (RD2)

As shown in Table 11, the models for acute bronchial and lower respiratory infections (RD2) were less reliable since the F ratio to test the overall goodness of fit of the regression equation was found significant in model 8 (O_3-IV) only. Variation in emergency room visits for RD2 explained by the regression equations fluctuated between 0% in model 4 and 39% in model 2.

Only models 2 and 3 exhibit a statistically significant coefficient

TABLE 10 MORBIDITY MODELS: ALLERGIC CONDITIONS AND UPPER RESPIRATORY INFECTIONS (RD1)

		(1) TSP-II	(2) SO_2-I	(3) SO_2-III	(4) NO_2-I	(5) NO_2-III	(6) CO-III	(7) NO-III	(8) O_3-IV
Multiple R		.54	.63	.58	.54	.57	.57	.59	.57
R^2		.29	.40	.33	.29	.33	.33	.34	.33
F ratio		2.98**	1.86	10.32***	1.53	9.98***	10.00***	9.25***	2.89*
d.f.		5,36	5,14	6,124	4,15	6,124	6,124	7,123	5,30
Constant		119.2	51.0	123.8	152.7	129.8	127.7	109.3	9.0
Pollutant	reg.	.183	1608	311	-887	11.32	1.01	161.5*	51.4
	s.e.	.181	1392	265	633	96.01	3.22	85.4	550.5
	F	(1.03)	(1.34)	(1.37)	(1.97)	(.01)	(.10)	(3.58)	(.01)
Temperature	reg.	-.652***		-.646***		-.712***	-.701***	-.633***	
	F	(8.57)		(30.07)		(40.06)	(42.12)	(31.88)	
Wind speed	reg.		4.61					1.14	
	F		(2.65)					(1.61)	
Humidity	reg.								
	F								
Precipitation	reg.	-17.62*	-27.07*	-11.53	-26.75*	-11.75	-11.62	-11.97	
	F	(3.08)	(4.05)	(2.46)	(3.95)	(2.53)	(2.47)	(2.66)	
Sky cover	reg.			1.13		1.20	1.21	1.15	5.84**
	F			(2.30)		(2.57)	(2.62)	(2.39)	(6.05)
Sunshine	reg.								.46
	F								(2.25)
Days-of-week effects									
Tuesday	reg.	3.69	-11.62	-.65	-15.5	-.67	-.69	-2.40	32.4**
	F	(.11)	(.39)	(.01)	(.50)	(.01)	(.01)	(.16)	(7.30)
Wednesday	reg.	1.30	.93	3.71	-2.87	3.81	3.84	4.01	34.9***
	F	(.01)	(.00)	(.39)	(.02)	(.41)	(.41)	(.46)	(7.91)

Note: 1) For each independent variable, regression coefficient, standard error (for pollutant only) and F statistic are represented on lines of reg., s.e. and F, respectively.
2) The level of significance is marked as asterisk: * (α=.10), ** (α=.05), and *** (α=.01)

TABLE 11 MORBIDITY MODELS: ACUTE BRONCHIAL AND LOWER RESPIRATORY INFECTIONS (RD2)

		(1) TSP-II	(2) SO_2-I	(3) SO_2-III	(4) NO_2-I	(5) NO_2-III	(6) CO-III	(7) NO-III	(8) O_3-IV
Multiple R		.42	.62	.27	.01	.22	.24	.24	.58
R^2		.18	.39	.08	.00	.05	.06	.06	.34
F ratio		1.54	1.37	2.04	.00	1.56	1.26	1.27	3.09**
d.f.		5,36	6,13	5,125	3,16	4,126	6,124	6,124	5,30
Constant		203.2	24.8	151.3	96.6	125.6	157.8	157.2	123.7
Pollutant	reg.	.122	5355**	-913**	.834	-155.8	.446	23.1	233.7
	s.e.	.220	2141	421	903	137.3	4.901	115.9	620.6
	F	(.31)	(6.26)	(4.70)	(.00)	(1.29)	(.01)	(.04)	(.14)
Temperature	reg.		1.01*	-.292*					
	F		(3.97)	(2.94)					
Wind speed	reg.		-6.78	-3.15		-2.41**	-1.91	-1.81	-7.05**
	F		(3.04)	(7.22)		(4.55)	(2.73)	(2.10)	(6.97)
Humidity	reg.	-1.46**					-.51		
	F	(6.61)					(1.88)		
Precipitation	reg.								
	F								
Sky cover	reg.		4.69						5.29**
	F		(2.79)						(5.24)
Sunshine	reg.	-.38*					-.18	-.18	
	F	(3.69)					(2.42)	(2.42)	
Days-of-week effects									
Tuesday	reg.	12.26	7.19	6.24	1.15	6.49	5.10	4.90	2.89
	F	(.72)	(.10)	(.57)	(.00)	(.60)	(.37)	(.33)	(.04)
Wednesday	reg.	5.80	37.37	10.24	.01	10.59	9.01	9.02	20.88
	F	(.15)	(2.13)	(1.49)	(.00)	(1.56)	(1.12)	(1.12)	(1.93)

Note: 1) For each independent variable, regression coefficient, standard error (for pollutant only) and F statistic are represented on lines of reg., s.e. and F, respectively.
2) The level of significance is marked as asterisk: * (α=.10), ** (α=.05), and *** (α=.01)

for the pollutant included (SO_2 in both cases). However, the coefficient of SO_2 in model 3 was negative. This is contradictory to the common knowledge that SO_2 is harmful to the respiratory tracts. Model 3 is not appropriate to estimate effects of SO_2 on RD2. Despite the small sample size in model 2 (of which the SO_2 variable was based on the average exposure level of patients), the standard error of SO_2, 2141, was less than half of its regression coefficient, 5355, with a significant F statistic of $6.26 > F(.05, 1, 13) = 4.67$. Other independent variables included in model 2 were average temperature, wind speed, sky cover and days-of-week.

MODELS FOR TOTAL CARDIAC DIAGNOSES (TCD)

Table 12 shows the regression models for total cardiac diagnoses (TCD). The variations of TCD explained by the equations were fairly small as compared with those of TRD in Table 9, except in model 2 which explains 48%. In short, R^2's were 12% in both models 3 and 6, 13% in 7, 24% in 1, 25% in 8, 26% in 4, and 48% in 2. Significant F ratios were produced in models 3, 5, 6 and 7. It is interesting that all models for TCD had a significant day-of-week effect to some extent, while only model 8 (O_3-N) for TRD did.

Three pollutants exhibited significant regression coefficients; SO_2 in model 2, NO_2 in 5, and NO in 7. In model 2, the regression coefficient and standard error of SO_2 were 3869 and 1707, respectively with a significant F value, $5.13 > (.05, 1, 13) = 4.67$. Wind speed, sky cover and sunshine were included in the model because they substantially improved the significance of the total model and the coefficient of SO_2. The days-of-week variables were significant (F values, 4.29 and $3.16 > F(.10, 1, 193) = 3.14$). Forty-eight per cent of the variation of TCD was explained in model 2, but its F ratio did not reach the significant level because of the small sample size. In model 5 (NO_2-III), the regression coefficient, standard error and F statistic of NO_2 were 224, 115 and 3.80 ($> F(.10, 1, 120) = 2.75$, or $< F(.05, 1, 120) = 3.92$), respectively. Only 14% of the variation of TCD was explained by this model which included sunshine, temperature and precipitation, in addition to the days-of-week variables. The overall goodness of fit of the model was significant, as indicated by its F ratio, $3.35 > F(.01, 6, 120) = 2.96$.

TABLE 12 MORBIDITY MODELS: TOTAL CARDIAC DIAGNOSES (TCD)

		(1) TSP-II	(2) SO_2-I	(3) SO_2-III	(4) NO_2-I	(5) NO_2-III	(6) CO-III	(7) NO-III	(8) O_3-IV
Multiple R		.49	.69	.34	.52	.37	.35	.36	.50
R^2		.24	.48	.12	.26	.14	.12	.13	.25
F ratio		2.29	2.01	3.25***	1.37	3.35***	2.93*	3.83***	1.97
d.f.		5,36	6,13	5,125	4,15	6,124	6,124	5,125	5,30
Constant		62.2	-14.2	74.2	179.0	109.2	85.3	102.1	18.3
Pollutant	reg.	.119	3869**	240	-859	224*	.602	155.9*	-891.7
	s.e.	.195	1707	288	976	115	3.887	81.81	791.1
	F	(.38)	(5.13)	(.70)	(.77)	(3.80)	(.02)	(3.63)	(1.27)
Temperature	reg.					-.198	-.175		
	F					(1.99)	(1.81)		
Wind speed	reg.		-3.03		-5.40				
	F		(1.07)		(1.64)				
Humidity	reg.								
	F								
Precipitation	reg.	-19.71*		-14.30		-12.21	-12.36	-12.38	
	F	(3.06)		(2.67)		(1.90)	(1.92)	(2.10)	
Sky cover	reg.	2.52*	8.90	2.88***			2.75***		5.75
	F	(2.98)	(1.99)	(10.29)			(9.21)		(2.84)
Sunshine	reg.		.77			-.25***		-.242***	.67
	F		(1.39)			(8.68)		(9.58)	(2.26)
Days-of-week effects									
Tuesday	reg.	27.5**	38.06*	11.34	29.01	9.89	11.3	9.59	25.76
	F	(5.14)	(4.29)	(2.54)	(1.39)	(1.95)	(2.53)	(1.83)	(2.23)
Wednesday	reg.	16.5	36.43*	9.25	12.39	7.75	9.32	8.58	36.65*
	F	(1.73)	(3.16)	(1.65)	(.37)	(1.17)	(1.68)	(1.45)	(4.22)

Note: 1) For each independent variable, regression coefficient, standard error (for pollutant only) and F statistic are represented on lines of reg., s.e. and F, respectively.
2) The level of significance is marked as asterisk: * (α=.10), ** (α=.05), and *** (α=.01)

Model 7 showed a significant association between NO and TCD. The pollutant's regression coefficient, standard error and F statistic were 155.9, 81.81 and 3.63 (> F (.10, 1, 120) = 2.75, or < F (.05, 1, 120) = 3.92), respectively. Only 13% of the variation of TCD was explained by this model which included sunshine, precipitation and the days-of-week variables. Its F ratio to test the goodness of fit of the model was 3.85, indicating statistical significance (i.e., 3.83 > F (.01, 5, 120) = 3.17).

MODELS FOR HYPERTENSION AND VASCULAR HEART DISEASE (CD1)

Table 13 is a list of regression models for hypertension and vascular heart disease (CD1). The variation of CD1 explained in each model ranged from 10% in model 3 to 46% in model 2. Significant F ratios appeared in models 2, 3, 5, 6, 7 and 8. Some models show significant day-of-week effects, but not as strong as TCD.

Two models indicated significant regression coefficients for the pollutants, but only one model, model 7, was valid since the other, model 8, showed a negative regression coefficient for O_3. This negative effect of O_3 on CD1 was caused by its inverse relationship with CD1 (-0.299), as provided in Table 18 in Appendix. Since O_3 would not reduce a risk of having CD1, this regression model was unacceptable for O_3-CD1 analysis. In model 7, standard error of NO, 94.4, was half the size of its regression coefficient, 183.3, with a significant F value, 3.77>F(.10, 1, 120) = 2.75, or < F(.05, 1, 120)= 3.92. Sunshine was chosen as an additional independent variable because it significantly improved the relationship between CD1 and NO. Although the coefficient of the pollutant in each of six other models did not attain the significance level of 10%, models 2 and 5 showed considerably smaller standard errors for SO_2 and NO_2, respectively, than their regression coefficients. In model 2, 46% of the variation of CD1 was explained by the regression equation including precipitation, sky cover and the days-of-week variables, in addition to the SO_2 variable (regression coefficient = 2206, standard error = 1581 and F value = 1.95<F(.10, 1, 14) = 3.10). The F ratio to test the overall goodness of fit of model 2 was 2.42 > F(.10, 5, 14) = 2.31. In model 5, only 13% of the variation was explained by the equation including sunshine,

TABLE 13 MORBIDITY MODELS: HYPERTENSION AND VASCULAR HEART DISEASE (CD1)

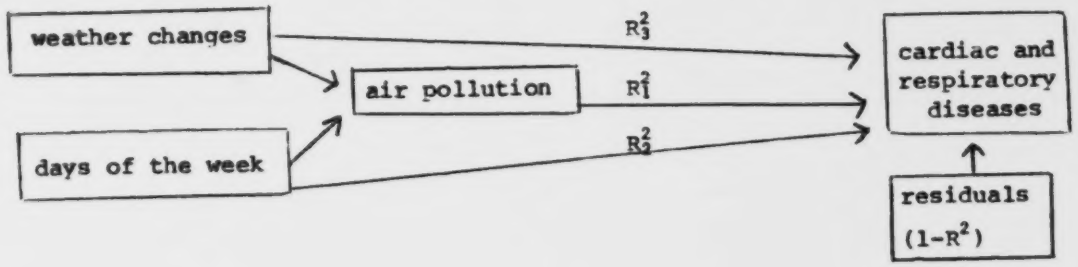
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
		TSP-II	SO_2-I	SO_2-III	NO_2-I	NO_2-III	CO-III	NO-III	O_3-IV
Multiple R		.45	.68	.32	.63	.36	.37	.35	.45
R^2		.20	.46	.10	.40	.13	.14	.13	.20
F ratio		1.80	2.42*	3.63***	1.83	3.02***	2.78**	4.52***	2.66*
d.f.		5.36	5,14	4,126	5,14	6,124	7,123	4,126	3,32
Constant		63.6	63.9	107.3	58.6	114.3	103.6	102.5	116.7
Pollutant	reg.	.107	2206	253	275.1	187.0	-5.67	183.3*	-1494**
	s.e.	.217	1581	335	742.6	134.4	4.83	94.4	692
	F	(.243)	(1.95)	(.57)	(.14)	(1.94)	(1.38)	(3.77)	(4.66)
Temperature	reg.					-.159			
	F					(.94)			
Wind speed	reg.						-1.46		
	F						(1.67)		
Humidity	reg.						.46		
	F						(1.50)		
Precipitation	reg.	-24.54*	-19.51		-24.3	-11.77	-16.36		
	F	(3.60)	(1.92)		(2.76)	(1.30)	(2.54)		
Sky cover	reg.	3.10*	2.42		3.11				
	F	(3.45)	(1.55)		(1.91)				
Sunshine	reg.			-.295***		-.329***	-.25**	-.29***	
	F			(10.97)		(11.27)	(4.79)	(11.03)	
Days-of-week effects									
Tuesday	reg.	21.17	29.69	7.56	38.7	5.77	7.53	6.09	10.18
	F	(2.33)	(2.51)	(.84)	(2.47)	(.49)	(.83)	(.56)	(.25)
Wednesday	reg.	14.37	-.23	9.51	.09	8.23	9.52	9.57	41.23*
	F	(.99)	(.00)	(1.29)	(.00)	(.97)	(1.30)	(1.34)	(4.03)

Note: 1) For each independent variable, regression coefficient, standard error (for pollutant only) and F statistic are represented on lines of reg., s.e. and F, respectively.
2) The level of significance is marked as asterisk: * (α=.10), ** (α=.05), and *** (α=.01)

precipitation, temperature and the days-of-week variables, in addition to the NO_2 variable (regression coefficient = 187.0, standard error = 134.4 and F value = 1.94 < F (.10, 1, 120) = 2.75). The F ratio was 3.02 > F (.01, 6, 120) = 2.96.

THE VARIATION DUE TO THE POLLUTANT, THE DAYS-OF-WEEK EFFECTS AND WEATHER CHANGES

The variation in each disease group explained by the regression model was divided into three portions; (1) due to the pollutant (R_1^2), (2) due to the days-of-week variables (R_2^2), (3) due to the climatological factors (R_3^2). Fourteen models were selected from Tables 9-13 according to F values or standard errors of the pollutants which were considered to be a significant or meaningful contribution to the regression model. Table 14 shows percentages of the variation due to the pollutant, the days-of-week variables and the climatological percentage factors, R^2 (sum of R_1^2, R_2^2 and R_3^2) and ($1-R^2$) which is the percentage of variance unexplained or attributable to the residuals. The following path diagram explains the possible relationship between cardiac and respiratory diseases and three factors:



R_1^2 (the variation due to the pollutant) included the variation shared by the dependent variable (the disease) and the pollutant without partitioning out the variation due to the days-of-week and weather variables, because weather changes have direct influences on air pollution levels which are also affected by human activities (i.e., industrial operations, traffic conditions, etc.) according to the days of the week.

In the column of R_1^2 (the variation due to the pollutant), model 2 (based on the average exposure levels of SO_2) showed the highest per cent of variation in the disease groups, TRD (13.28%), TCD (21.72%), and CD1

TABLE 14

THE VARIATION DUE TO THE POLLUTANT, DAYS-OF-WEEK EFFECTS AND WEATHER CHANGES IN THE MODELS SELECTED FROM TABLES 9-13

Disease	Model	Total Variation (%) Due to Pollutant (R_1^2)	Due to days-of-week effects (R_2^2)	Due to weather changes (R_3^2)	R^2	$1-R^2$
TRD	(1) TSP-II	0.29	1.14	25.58	27.01	72.99
	(2) SO_2-I	13.28	8.14	14.20	35.62	64.38
	(6) CO-III	3.35	0.57	28.89	32.81	67.19
	(7) NO-III	7.60	0.73	24.43	32.76	67.24
RD1	(2) SO_2-I	9.62	8.05	22.21	39.88	60.12
	(3) SO_2-III	11.69	0.30	21.31	33.30	66.70
	(7) NO-III	6.50	0.55	27.43	34.48	65.52
RD2	(2) SO_2-I	13.45	0.77	24.50	38.72	61.28
TCD	(2) SO_2-I	21.72	12.82	13.55	48.09	51.91
	(5) NO_2-III	0.62	2.37	10.94	13.93	86.07
	(7) NO-III	4.36	2.01	6.92	13.29	86.71
CD1	(2) SO_2-I	21.48	14.44	10.48	46.40	53.60
	(5) NO_2-III	0.12	1.19	11.45	12.76	87.24
	(7) NO-III	3.84	1.05	7.65	12.54	87.46

(21.48%). In RD1, the variation due to SO_2 from the University of Illinois Medical Center in model 3, 11.69%, was greater than that based on the average exposure levels in model 2, 9.62%. In RD2, only model 2 was chosen and SO_2 explained 13.45% of the variance. TSP accounted for only 0.29% of the variation in TRD. CO showed 3.35% of the variation in TRD. The variation due to NO was 7.60% in TRD, 6.50% in RD1, 4.36% in TCD and 3.84% in CD1. The variation due to NO_2 was very small, 0.62 in TCD and 0.12% in CD1.

The variation due to the days-of-week variables (R_2^2) was small in all models except in model 2 which might be due to its small sample size. The variation due to weather changes (R_3^2) ranged from 6.92% to 28.89%. R_3^2 was greater than R_1^2 in all models except model 2 for TCD and for CD1.

COMMENTS

In the previous study by Namekata and Carnow[27], the regression model for all respiratory conditions were shown as follows:

% of excess visits = 90.24 + 557.42 SO_2 - 0.389 Max temp. + 1.797 Windspeed

(326.69) (0.188) (0.945)

2.91 4.27 3.62

where % of excess visits was the same definition as the one in the present study. SO_2 was the average patient exposure level (ppm), and max temp. and wind speed were daily maximum temperature (^{o}F) and daily average wind speed (miles per hour), respectively. Figures under each coefficient indicate a standard error in parentheses and an F value with an underline. The days-of-the-week variables were not included because only Tuesday's data were used. Thirty-six per cent of the variation of this disease category was explained by the above model, while model 2(SO_2-I) for total respiratory diagnoses in Table 9 had also a R^2 of 0.36. SO_2 in the above model was significant at the .10 level while SO_2 in model 2 in Table 9 approached significance. Although the previous study used data from 14 hospitals in Chicago, the strength of the relationship between SO_2 and respiratory disease was similar to the one in the present study. Despite the limited data base this similarity implies that sulfur dioxide might have acute effects on respiratory disease at fairly low levels which are usually found in the Chicago area.

Another important finding in the previous study[27] was the significant relationship between total suspended particulate (the daily average of patient exposure levels) and bronchitis, as shown in the following model:

% of excess visits=-5.51+0.404TSP-1.234 min.temp.+5.490 wind speed+0.845 Humidity

(0.203) (0.273) (1.585) (0.429)

3.95 20.40 12.00 3.875

where % of excess visits for bronchitis was the same as the one defined before, TSP was the average patient exposure level (ug/m^3), and min.temp., wind speed and humidity were daily measurements of minimum temperature (^{o}F), wind speed (miles per hour) and humidity (%), respectively. Figures of each independent variable were defined in the model for all respiratory conditions. The coefficient of TSP was significant at the .10 level and close to the .05 level ($3.95 > F(.10,1,40)=2.84$, but $3.95 < F(.05,1,40)=4.08$). Forty-seven per cent

of the variation of bronchitis patient visits was explained by this model. A comparison was not possible between this model and model 1 (TSP-II) in Table 11 - models for acute bronchial and lower respiratory infections (RD2) in the present study, because the disease group for RD2 consisted of nine respiratory diagnoses; asthma, bronchial astham, bronchitis, acute bronchitis, tracheobronchitis, pleurisy, pleuritis, broncheolitis and pneumonia. The insignificant association between TSP and RD2 in the present study might be caused by this disease classification, since the regression model for asthma in the previous study did not show a significant association with either SO_2 or TSP, while the model for bronchitis had a significant coefficient of TSP as described above.

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APPENDIX

TABLE 15 CORRELATION COEFFICIENTS BETWEEN EMERGENCY ROOM VISITS FOR CARDIAC AND RESPIRATORY CONDITIONS AND ENVIRONMENTAL MEASUREMENTS IN PHASE I ANALYSIS

	(1) Total respiratory diagnoses	(2) Allergic conditions and upper respiratory infections	(3) Acute bronchial and lower respiratory infections	(4) Total Cardiac Diagnoses	(5) Hypertension and vascular Heart Disease
TSP-CN	-0.028	-0.121	0.045	-0.011	-0.074
SO_2-CN	0.364	0.319	0.367	0.466	0.463
NO_2-CN	-0.172	-0.260	-0.005	-0.229	-0.248
SO_2-Polk St.	-0.035	-0.053	0.124	0.242	0.233
NO_2-Polk St.	0.011	-0.142	0.114	0.056	0.027
CO- Polk St.	-0.146	-0.168	-0.004	-0.134	-0.155
NO- Polk St.	0.003	-0.079	0.248	0.273	0.278
Temperature	-0.192	-0.219	0.072	-0.212	-0.161
Wind speed	0.196	0.306	-0.148	-0.126	-0.137
Humidity	0.143	0.152	-0.101	0.051	0.110
Precipitation	-0.348	-0.426	0.024	-0.301	-0.303
Sky cover	0.199	-0.020	0.103	0.151	0.242
Sunshine	-0.193	0.122	-0.090	-0.133	-0.199

Note: Sample size N=20

Critical values of the correlation coefficient: 0.444 (α=.05), 0.561 (α=.01)

CN after the pollutants indicates the measurements from the city air sampling network which were used to estimate the patient exposure levels.

Polk St. after the pollutants indicates the measurements from the Illinois E.P.A. sampling station on Polk Street at the University of Illinois Medical Center.

TABLE 16 CORRELATION COEFFICIENTS BETWEEN EMERGENCY ROOM VISITS FOR CARDIAC AND RESPIRATORY CONDITIONS AND ENVIRONMENTAL MEASUREMENTS IN PHASE II ANALYSIS

	(1)	(2)	(3)	(4)	(5)
	Total Respiratory Diagnoses	Allergic Conditions and upper respiratory infections	Acute bronchial and lower respiratory infections	Total cardiac diagnoses	hypertension and vascular heart disease
TSP-CN	0.054	-0.010	0.070	-0.011	0.014
SO_2-Polk St.	0.210	0.283	0.011	0.242	0.256
NO_2-Polk St.	-0.041	-0.144	0.072	0.056	-0.023
CO- Polk St.	0.110	0.084	0.053	-0.134	-0.120
NO- Polk St.	0.209	0.190	0.119	0.273	0.213
Temperature	-0.407	-0.448	0.001	-0.212	-0.117
Wind speed	-0.016	-0.006	-0.111	0.126	-0.010
Humidity	0.038	0.119	-0.266	0.051	0.146
Precipitation	-0.323	-0.345	-0.069	-0.301	-0.216
Sky cover	0.052	0.019	0.062	0.151	0.214
Sunshine	-0.131	-0.108	-0.061	-0.133	-0.204

Note: Sample size N=42

Critical values of the correlation coefficient; .304 (α=.05), .393 (α=.01)

CN after the pollutants indicates the measurements from the city air sampling network which were used to estimate the patient exposure levels.

Polk St. after the pollutants indicates the measurements from the Illinois E.P.A. sampling station on Polk Street at the University of Illinois Medical Center.

TABLE 17 CORRELATION COEFFICIENTS BETWEEN EMERGENCY ROOM VISITS FOR CARDIAC AND RESPIRATORY CONDITIONS AND ENVIRONMENTAL MEASUREMENTS IN PHASE III ANALYSIS

	(1) Total respiratory diagnoses	(2) Allergic conditions and upper respiratory infections	(3) Acute bronchial and lower respiratory infections	(4) Total cardiac diagnoses	(5) Hypertension and vascular heart disease
SO_2- Polk St.	0.294	0.342	-0.055	0.123	0.117
NO_2- Polk St.	-0.128	-0.183	-0.028	0.078	0.035
CO- Polk St.	0.183	0.129	0.065	0.040	-0.028
NO- Polk St.	0.276	0.255	0.094	0.209	0.196
Temperature	-0.552	-0.549	-0.049	-0.185	-0.168
Wind speed	-0.050	0.030	-0.163	-0.095	-0.103
Humidity	0.170	0.145	-0.039	0.176	0.240
Precipitation	-0.155	-0.176	-0.009	-0.078	-0.037
Sky cover	0.180	0.157	0.058	0.253	0.278
Sunshine	-0.255	-0.217	-0.086	-0.259	-0.208

Note: Sample size N=131

Critical values of the correlation coefficient in df = 100; .195 (α=.05), .254 (α=.01)

CN after the pollutants indicates the measurements from the city air sampling network which were used to estimate the patient exposure levels.

Polk St. after the pollutants indicates the measurements from the Illinois E.P.A. sampling station on Polk Street at the University of Illinois Medical Center.

TABLE 18 CORRELATION COEFFICIENTS BETWEEN EMERGENCY ROOM VISITS FOR CARDIAC AND RESPIRATORY CONDITIONS AND ENVIRONMENTAL MEASUREMENTS IN PHASE IV ANALYSIS

	(1) Total respiratory diagnoses	(2) Allergic conditions and upper respiratory infections	(3) Acute bronchial and lower respiratory infections	(4) Total cardiac diagnoses	(5) Hypertension and vascular heart disease
O_3 - Polk St.	-0.147	-0.116	-0.215	-0.247	-0.299
Temperature	-0.131	-0.069	-0.238	-0.163	-0.264
Wind speed	-0.253	-0.089	-0.407	-0.202	-0.136
Humidity	0.290	0.213	0.214	0.104	0.132
Precipitation	-0.016	-0.091	0.121	0.001	0.024
Sky cover	0.261	0.172	0.301	0.180	0.162
Sunshine	-0.080	0.019	-0.233	-0.025	-0.057

Note: Sample size N=36

Critical values of the correlation coefficient; .330 (α=.05), .424 (α=.01)

CN after the pollutants indicates the measurements from the city air sampling network which were used to estimate the patient exposure levels.

Polk St. after the pollutants indicates the measurements from the Illinois E.P.A. sampling station on Polk Street at the University of Illinois Medical Center.

TABLE 19 CORRELATION MATRIX OF ENVIRONMENTAL MEASUREMENTS IN PHASE I ANALYSIS

	TSP-CN	SO_2-CN	NO_2-CN	SO_2-Polk	NO_2-Polk	CO-Polk	NO-Polk	Temp.	Wind speed	Humidity	Precipitation	Sky-cover	Sunshine
TSP-CN	1.000	0.076	0.536	-0.047	0.360	0.580	0.179	0.426	-0.385	-0.725	-0.171	-0.603	0.667
SO_2-CN	0.076	1.000	-0.013	0.477	-0.015	0.163	0.093	-0.417	0.075	0.125	-0.145	0.154	-0.259
NO_2-CN	0.536	-0.013	1.000	0.427	0.179	0.512	0.429	-0.034	-0.679	-0.548	-0.001	-0.296	0.342
SO_2-MD	-0.047	0.477	0.427	1.000	-0.206	0.467	0.182	-0.646	-0.441	-0.152	-0.132	0.031	-0.144
NO_2-MD	0.360	-0.015	0.179	-0.206	1.000	-0.032	0.541	0.340	-0.386	-0.214	-0.067	-0.133	0.193
CO-MD	0.580	0.163	0.512	0.467	-0.032	1.000	0.191	-0.082	-0.403	-0.379	-0.316	-0.345	0.326
NO-MD	0.179	0.093	0.429	0.182	0.541	0.191	1.000	-0.148	-0.614	0.123	-0.182	0.102	0.064
Temp.	0.426	-0.417	-0.034	-0.646	0.340	-0.082	-0.148	1.000	0.120	-0.432	0.170	-0.388	0.509
Wind speed	-0.385	0.075	-0.679	-0.441	-0.386	-0.403	-0.614	0.120	1.000	0.399	0.181	0.224	-0.305
Humidity	-0.725	0.125	-0.548	-0.152	-0.214	-0.379	0.123	-0.432	0.399	1.000	0.136	0.780	-0.767
Precipitation	-0.171	-0.145	-0.001	-0.132	-0.067	-0.316	-0.182	0.170	0.181	0.136	1.000	0.277	-0.288
Sky cover	-0.603	0.154	-0.296	0.031	-0.133	-0.345	0.102	-0.388	0.224	0.780	0.277	1.000	-0.936
Sunshine	0.667	-0.259	0.342	-0.144	0.193	0.326	0.064	0.509	-0.305	-0.767	-0.288	-0.936	1.000

Note: Sample size N=20

Critical values of the correlation coefficient; 0.444 (α=.05), 0.561 (α=.01)

TABLE 20 CORRELATION MATRIX OF ENVIRONMENTAL MEASUREMENTS IN PHASE II ANALYSIS

	TSP-CN	SO_2-Polk	NO_2-Polk	CO-Polk	NO-Polk	Temp.	Wind speed	Humidity	Precipitation	Sky cover	Sunshine
TSP-CN	1.000	0.266	0.404	0.501	0.434	0.366	-0.565	-0.216	-0.013	-0.228	0.246
SO_2-MD	0.266	1.000	-0.093	0.454	0.575	-0.468	-0.511	0.119	-0.035	0.134	-0.294
NO_2-MD	0.404	-0.093	1.000	0.220	0.333	0.332	-0.376	0.129	0.106	-0.022	0.104
CO-MD	0.501	0.454	0.220	1.000	0.458	-0.173	-0.406	-0.058	-0.063	-0.218	0.006
NO-MD	0.434	0.575	0.333	0.458	1.000	-0.215	-0.662	0.114	-0.047	0.099	-0.146
Temp.	0.366	-0.468	0.332	-0.173	-0.215	1.000	-0.096	-0.176	0.200	-0.032	0.223
Wind speed	-0.565	-0.511	-0.376	-0.406	-0.662	-0.096	1.000	0.210	0.050	0.037	-0.026
Humidity	-0.216	0.119	0.129	-0.058	0.114	-0.176	0.210	1.000	0.279	0.628	-0.615
Precipitation	-0.013	-0.035	0.106	-0.063	-0.047	0.200	0.050	0.279	1.000	0.344	-0.321
Sky cover	-0.228	0.134	-0.022	-0.218	0.099	-0.032	0.037	0.628	0.344	1.000	-0.888
Sunshine	0.246	-0.294	0.104	0.006	-0.146	0.223	-0.026	-0.615	-0.321	-0.888	1.000

Note: Sample size N=42

Critical values of the correlation coeffient: .304 (α=.05), .393 (α=.01)

TABLE 21 CORRELATION MATRIX OF ENVIRONMENTAL MEASUREMENTS IN PHASE III ANALYSIS

	SO_2-Polk	NO_2-Polk	CO-Polk	NO-Polk	Temp.	Wind speed	Humidity	Precipitation	Sky cover	Sunshine
SO_2-MD	1.000	-0.006	0.468	0.643	-0.469	-0.377	0.111	-0.081	-0.127	-0.175
NO_2-MD	-0.006	1.000	0.261	0.344	0.347	-0.363	0.045	0.046	-0.080	0.182
CO-MD	0.468	0.261	1.000	0.568	-0.198	-0.398	0.071	-0.117	-0.063	-0.018
NO-MD	0.643	0.344	0.568	1.000	-0.269	-0.526	0.163	-0.073	0.110	-0.108
Temp.	-0.469	0.347	-0.198	-0.269	1.000	-0.060	-0.135	0.170	-0.139	0.313
Wind speed	-0.377	-0.363	-0.398	-0.526	-0.060	1.000	-0.057	0.052	-0.046	0.048
Humidity	0.111	0.045	0.071	0.163	-0.135	-0.057	1.000	0.327	0.595	-0.641
Precipitation	-0.081	0.046	-0.117	-0.073	0.170	0.052	0.327	1.000	0.299	-0.261
Sky cover	0.127	-0.080	-0.063	0.110	-0.139	-0.046	0.595	0.299	1.000	-0.881
Sunshine	-0.175	0.182	-0.018	-0.108	0.313	0.048	-0.641	-0.261	-0.881	1.000

Note: Sample size N=131

Critical values of the correlation coefficient in df = 100, .195 (α=.05), .254 (α=.01)

TABLE 22 CORRELATION MATRIX OF ENVIRONMENTAL MEASUREMENTS IN PHASE IV ANALYSIS

	O_3-Polk	Temperature	Wind speed	Humidity	Precipitation	Sky cover	Sunshine
O_3- Polk	1.000	0.619	0.101	-0.356	-0.309	-0.626	0.670
Temperature	0.619	1.000	0.244	-0.016	-0.027	-0.343	0.287
Wind speed	0.101	0.244	1.000	-0.076	0.267	-0.018	-0.066
Humidity	-0.356	-0.016	-0.076	1.000	0.415	0.494	-0.701
Precipitation	-0.309	-0.027	0.267	0.415	1.000	0.405	-0.478
Sky cover	-0.626	-0.343	-0.018	0.494	0.405	1.000	-0.789
Sunshine	0.670	0.287	-0.066	-0.701	-0.478	-0.789	1.000

Note: Sample size N=36

Critical values of the correlation coefficient; .330 (α=.05), .424 (α=.01)

Table 23. Distribution by Race of Study Population; April, 1977 - April, 1978

Race	CCH Absolute Frequency	CCH Relative Frequency (%)	UIH Absolute Frequency	UIH Relative Frequency (%)	Total Absolute Frequency	Total Relative Frequency (%)
White	921	5.9	1275	27.3	2196	10.8
Black	14413	92.4	3217	68.9	17630	86.9
Other	260	1.7	144	3.1	404	2.0
Unspec.	30	0.2	32	0.7	62	0.3
	15624	100.0	4668	100.0	20292	100.0

Table 24. Distribution by Sex of Study Population; April, 1977 - April, 1978

Sex	CCH Absolute Frequency	CCH Relative Frequency (%)	UIH Absolute Frequency	UIH Relative Frequency (%)	Total Absolute Frequency	Total Relative Frequency (%)
Male	7315	46.8	2163	46.3	9478	46.7
Female	8286	53.0	2491	53.4	10777	53.1
Unspec.	23	0.2	14	0.3	37	0.2
	15624	100.0	4668	100.0	20292	100.0

TABLE 25. FREQUENCY AGE DISTRIBUTION OF TOTAL STUDY POPULATION, CCH AND UIH, April, 1977 - April, 1978

Age Category	CCH Absolute Frequency	CCH Relative Frequency (%)	UIH Absolute Frequency	UIH Relative Frequency (%)	Total Absolute Frequency	Total Relative Frequency (%)
5	29	0.2	1474	31.6	1503	7.4
5 - 9	15	0.1	491	10.5	506	2.5
10 - 14	13	0.1	265	5.7	278	1.4
15 - 19	2351	15.1	351	7.5	2702	13.3
20 - 24	2921	18.7	456	9.8	3377	16.6
25 - 29	2065	13.2	309	6.6	2374	11.7
30 - 34	1428	9.1	207	4.4	1635	8.1
35 - 39	1209	7.7	160	3.4	1369	6.7
40 - 44	1203	7.7	145	3.1	1348	6.6
45 - 49	1064	6.8	162	3.5	1226	6.0
50 - 54	979	6.3	138	3.0	1117	5.5
55 - 59	683	4.4	109	2.3	792	3.9
60 - 64	532	3.4	113	2.4	645	3.2
65 - 69	419	2.7	103	2.2	522	2.6
70 - 74	307	2.0	85	1.8	392	2.0
75+	317	2.0	85	1.8	402	2.0
#99 (unspec.)	89	0.6	15	0.3	104	0.5
	15624	100.0	4668	99.9	20292	100.0

Table 26 . Frequency of all Diagnostic Illnesses of Study Population April, 1977 - April, 1978

Diagnostic Group	Absolute Frequency	Relative Frequency (%)
Respiratory	13985	68.9
Cardiac	3294	16.2
Omissions	3013	14.9
	20292	100.0